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ABSTRACT

Nutrition is well-recognized as a necessary component of educational programs for physicians. This is to be valued in that of all factors affecting health in the United States, none is more important than nutrition. This can be argued from various perspectives, including health promotion, disease prevention, and therapeutic management. In all cases, serious consideration of nutrition related issues in the practice is seen to be one means to achieve cost-effective medical care. These modules were developed to provide more practical knowledge for health care providers, and in particular primary care physicians. This module is designed to encourage the primary care physician to stress to patients that osteoporosis is avoidable and to provide instruction on how patients can change their dietary habits. Answers are provided to commonly asked questions regarding bone and tooth development, the prevention of disease, and the role of nutrition in the maintenance of healthy bones and teeth. Included are learning goals and objectives, a self-check of achievement with regard to goals, references, and lists of resources for patients and physicians. (CW)

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24. **Behaviorial and Neurological Disorders**
25. **Preventing Hospital and Home Malnutrition**
26. **Questions About Common Ailments**

Faculty Guide (includes comprehensive index for Modules 1-26)

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21 Nutrition in Health Promotion: Protecting Bone and Teeth

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Introduction

Osteoporosis is a severe, often crippling, disease of old age that can deny your patients a happy, productive life in their later years. As many as 30,000 premature deaths per year in the U.S. are due to the primary or secondary effects of osteoporosis. The annual cost has been estimated to be $3.8 billion, a figure which is likely to climb substantially as the result of rapid increases in the geriatric population.[1]

Although relatively little can be done to reverse osteoporosis once there is an onset of fractures, there can be a great deal accomplished by focusing on the preventive measures effective against this debilitating disease.

For a review, sections on the processes which regulate bone metabolism have been included. Throughout this module, an attempt has been made to relate the clinical processes to the underlying physiological and biochemical processes involved in bone metabolism.

Goal

The goal of this module is to encourage the primary care physician to stress to patients that osteoporosis is avoidable; however, measures such as adequate daily calcium intake, exercise, and rational drug use need to be instituted as a matter of daily habit early in life rather than as a short-term treatment once a problem has been diagnosed. Answers will be provided to commonly asked questions regarding bone and teeth development, prevention of osteoporosis and dental caries, and the role of nutrition/diet in maintenance of healthy bones and teeth.

Objectives

Upon completion of this module, you will be able to:

1. *Discuss normal metabolism and pathological processes of bones and teeth.*
2. *Identify which population groups are at greatest risk for osteoporosis.*
3. *Recognize the need for calcium at various stages of the life cycle and distinguish between the needs of males and females.*
4. *Determine when preventive measures should be started to preserve teeth and bones.*
5. *Identify which dietary, drug, and exercise measures are most effective in maintaining teeth and bones.*
6. *Educate patients as to the factors which contribute to the good health of bones and teeth.*
7. *Counsel patients as to how to obtain the proper amount of calcium in the diet.*
8. *Determine which drugs, diseases, and treatment regimens exacerbate problems with bones and teeth.*

Definition of Osteoporosis and the Population at Risk.

Osteoporosis is the loss of bone mass which results in an increased number of fractures, primarily of the spine, hips, and forearms.

Osteoporosis is the most common skeletal disorder in the world and is characterized by a lowered bone mass, with the remaining bone normally mineralized. Although the osteoporotic process may be slowed or stopped, the existent damage is irreversible. This malady, osteopenia, is one of four subcategories of the loss of bone mass; the other three subcategories are osteomalacia, endocrinopathies, and marrow packing disorders, some of which are reversible.

The decrease in bone mass due to osteoporosis leads to an increased number of fractures, with the spine, hips, and forearms being most susceptible. Spinal compression fractures after such minor trauma as stepping down from a step, coughing, or receiving an enthusiastic hug are common. Different subgroups of osteoporotic individuals may have greater or lesser incidence of fractures at various bone sites.

Osteoporosis not only has an extreme economic impact, but it also can affect the overall general health of its victims. The same factors which maintain good bone health are also responsible for maintenance of sound teeth.

In the U.S. alone, osteoporosis is implicated in nearly 200,000 hip fractures and 100,000 wrist fractures per year, affecting predominantly women, in particular about 15-25% of all postmenopausal women. In the spine, the lower thoracic and upper lumbar regions are most affected. In addition to being painful and temporarily debilitating, these types of fractures can have long-term consequences (Table 21-1).

The lowered thoracic and abdominal volume may lower appetite and decrease lung volume, thereby impairing ability to exercise. The yearly costs in the U.S. from the acute problems of osteoporosis alone are staggering, in the order of billions of dollars.[2] This figure does not include requirements for chronic care, long-term disability, or lowered quality of life.

Table 21-1. Consequences of Osteoporosis

1. Reduced stature
2. Dowager's hump
3. Alteration of rib position leading to
   a. Lowered thoracic volume
   b. Lowered abdominal volume

Although osteoporosis does not include strictly dental problems such as peridontal disease, the same factors which help to maintain strong bones also affect the soundness of teeth. The calcified tissues in teeth are the enamel, dentin, and cementum. These tissues are more influenced by external factors during the developmental period than after the teeth have erupted. When bones of the jaw deteriorate, however, teeth may loosen and eventually be lost. Thus, throughout this discussion it should be borne in mind that the discussion also applies to teeth. Because of the pervasiveness of osteoporosis, it is important to understand the mechanisms for the development of the disease and the preventive measures which may be taken to reduce or eliminate the problem.

Susceptibility to osteoporosis may be related to peak bone mass which is attained at approximately age 35 in both sexes.

Bone mineral mass peaks at roughly age 35 for both sexes and declines at a greater rate for females than for males; it is particularly accelerated after menopause. The major determinant for osteoporosis in old age may be the peak bone mass attained early in adult life.[3] Therefore, measures aimed at preventing osteoporosis should be directed not only at the older segments of the population but also at children and young adults who have not yet attained peak bone mass. Factors affecting peak bone mass will be discussed in various sections of this module.

Bone Composition and Structure

Bones are 65% mineral, with a calcium and phosphorus crystal type called hydroxyapatite being the major component. There are two

types of bone in the skeleton, tubular and cancellous, and it is the cancellous type which is subject to osteoporosis.

Bone is composed of minerals, water, and an organic matrix consisting of 95% collagen and 5% extracellular fluid and mucoprotein. The mineral component of bone (65% of total weight) is composed mostly of hydroxyapatite, $CA_{10}(PO_4)_6(OH)_2$; 30-40% amorphous calcium phosphate; and small amounts of other crystalline types like octacalcium phosphate, $CA_8H_2(PO_4)\bullet H_20_5$. Of the approximately 600 gm of phosphorus in the adult human, 85% of it is in the skeleton. Teeth contain similar types of crystals, but the percentages of each type vary in the three kinds of calcified tissue of the teeth.

The skeleton has two kinds of bone—tubular and cancellous. Tubular bone has a cortex and a few underlying trabeculae (bone spicules or the fibrous framework) and is found predominantly in the appendicular skeleton. Cancellous or spongy bone, on the other hand, has a thin cortex; numerous trabeculae provide support. Cancellous bone is found mostly in the axial skeleton in the spine, in the head and neck of the femur, and in the distal radius. It is the cancellous bone which is preferentially subject to osteoporosis. Radiologists can quantitate the degree of bone loss by the Singh index, which is based upon the disappearance of trabeculae from the neck and head of the femur.[4] However, up to 30% bone loss may occur before overt losses can be detected radiographically.

Tooth formation begins *in utero*, with calcification beginning at 5 months of gestation. The final stages of tooth development occur in the early twenties with the eruption of the wisdom teeth.

A number of factors can influence the health of a tooth long before the tooth actually erupts above the gum line. The tooth buds form *in utero* at 4-6 weeks of gestation. Calcification of the teeth begins at 5 months of gestation. After birth, the permanent teeth begin to calcify as early as 4-6 months of age, even though the eruption of these teeth does not occur until approximately 6 years of age; the process continues until the early twenties with the eruption of the wisdom teeth.

Proper tooth development requires adequate dietary intake of calcium, phosphorus, protein, and other essential minerals. Vitamin A deficiency *in utero* may result in inadequately developed tooth enamel, a circumstance which leaves teeth more susceptible to decay later. Vitamin D deficiency delays tooth eruption and results in pitted, thin, or absent tooth enamel. Vitamin C deficiency impairs collagen formation and compromises the health of the gums.

Tooth decay (dental caries) occurs when bacteria metabolize food stuck to the surfaces of the teeth producing organic acids which destroy the tooth enamel. A newly erupted tooth has the greatest susceptibility to development of caries. The frequency of caries can be reduced by

1. brushing teeth after eating and using floss daily,
2. avoiding sugar-containing snacks unless tooth brushing follows promptly,
3. avoiding sticky foods such as caramels which hold carbohydrate-containing foods next to the teeth,
4. rinsing the mouth with warm water if brushing cannot be accomplished immediately,
5. drinking fluoridated water or using a fluoridated rinse with the advice of a dentist, and
6. having regular dental check-ups and professional tooth cleaning.

Another problem that can affect teeth in young children is the "nursing bottle" syndrome. Persons who put their children to bed with a bottle of milk or fruit juice may encounter this problem which leads to massive decay of the upper front teeth. As the child falls asleep, saliva production ceases and the milk or juice pools around the front top teeth. Bacterial growth with resultant acid production then occurs, promoting caries in the upper teeth. The lower teeth, protected by the tongue, are seldom involved. The practice of putting infants to bed with a bottle should be discouraged. Further, soft drinks or other high-sugar drinks should not constitute part of a diet for an infant.

Osteoblasts are involved in bone formation; osteoclasts are active in bone resorption; and osteocytes are resting bone cells embedded in the mineral matrix.

In order to understand the factors that result in formation and maintenance of strong, fracture-resistant

bones, it is first necessary to examine processes involved in metabolic bone formation. An excellent two-part review has appeared recently.[5,6] The process will be examined only briefly here.

The major functioning cells of bone (osteoblasts, osteoclasts, and osteocytes) originate from a common mesenchymal stem cell. Their functions are summarized in Table 21-2.

Together osteoblasts and osteoclasts constitute 95% of the bone cells. Osteoblasts have several important roles in bone formation. Procollagen is synthesized and undergoes hydroxylation and glycosylation in the osteoblasts. After secretion from the osteoblasts, procollagen is attacked by a protease to form collagen which is then formed into fibrils stabilized by intra- and intermolecular crosslinks. Mineralization can then occur. Osteoblasts are also responsible for synthesizing other proteins important to the bone matrix, such as a calcium-binding protein, osteocalcin (whose synthesis is vitamin K- and vitamin D-dependent), and osteonectin (a phosphoprotein capable of crosslinking collagen and calcium). As new bone matrix is built, the osteoblasts become embedded in the bone and become resting cells or osteocytes. The final type of cells important to bone strength are the osteoclasts; these are involved in bone resorption. They are multinucleated cells which secrete proteolytic enzymes and organic acids (citrate and lactate) which are involved in the breakdown of existing bone. Teeth, of course, have the added insult of organic acids, such as those produced by bacteria in plaque, which erode the teeth from the surface.

Bone synthesis and remodelling are constant processes.

Through cell processes, communication among bone cells is maintained. There are two modes of osteogenesis: endochondral, wherein new bone develops from previously formed cartilage; and membranous, wherein new bone arises from connective tissue rather than cartilage. A reasonably constant bone mass is maintained by carefully balancing the relative activities of osteoblasts and osteoclasts. It is important to remember that bone is a living tissue which is constantly synthesized and remodelled.

Regulation of Bone Formation and Remodelling

A number of regulatory factors are involved in the overall control of bone formation and remodelling. The types of factors involved are listed in Table 21-3. Mechanisms must also exist to mediate effects of exercise and stress on bone. How these are mediated, however, is not at all clear.

One of the body's very crucial needs is to maintain serum calcium levels within a narrow range. As the major pool of calcium, bones play an active role in the maintenance of serum calcium levels.

Although bone growth and remodelling are important to structural integrity, the larger and more important role is that of providing calcium homeostasis.

Serum calcium levels are tightly maintained between 9 and 11 mg% (4.5-5.5 mEq/l) and excursions in either direction result in death. Therefore, it is necessary for the body to have a multiplicity of acute and chronic regula-

Table 21-2. Formation and Degradation of Bone

Cell Type	Function	Involvement
Osteoblasts	Bone formation	Synthesize procollagen Synthesize bone matrix proteins
Osteocytes	Resting cells	Osteoblasts which have become embedded in bone matrix
Osteoclasts	Bone degradation	Secrete proteolytic enzymes Secrete organic acids

Table 21-3. Regulators of Bone Formation and Remodelling	
I.	Factors in calcium homeostasis
	1. Parathyroid hormone
	2. Calcitonin
	3. Vitamin D
	4. Intracellular calcium regulators
II.	Hormones involved in nutrient homeostasis
	1. Insulin
	2. Growth hormone
	3. Thyroxine
	4. Sex steroids
	5. Glucocorticoids
III.	Factors which operate locally
	1. Prostaglandins
	2. Osteoclast-activating factor
	3. Bone-derived growth factor

tory mechanisms to ensure that bone, which is the major pool of body calcium (99% or 1200-1400 gm in a typical adult human), is maintained at a reasonably constant level. Half of the calcium in blood is present as "free" calcium (1mM), and half is bound to small molecules or plasma proteins. Calcium plays a role in muscle contraction, nerve conduction, and hormone secretion by coupling an appropriate stimulus with calcium mobilization in the secreting cell and intracellular communication; therefore, it is necessary that regulation of changes in the mineral composition of bone be intimately connected to the physiological processes in the rest of the body.

Because maintenance of stable calcium levels is so crucial to life, it seems reasonable that a number of regulatory processes and tissues should be involved in the overall control of calcium metabolism. Intake and excretion of calcium contribute to overall calcium balance, and there is considerable turnover of calcium throughout the body (calcium in extracellular fluid turns over between 40 and 50 times a day). In liver and heart the turnover is six times a day; in skeletal muscle the turnover is twice a year. While 50-60 gm of calcium in bone is readily exchangeable, the total bone turnover of calcium is on the order of 3%/year. Clearly, if the turnover process is not balanced, a significant loss of bone minerals can occur, even in as short a period as 10 years. An overall description of the regulation of calcium levels is shown in Table 21-4.

Table 21-4. Factors Regulating Serum Calcium		
Factors	Tissue	Effect*
Intake Versus Excretion		
Intestinal uptake	Intestine	+
Fecal excretion	Intestine	–
Renal excretion	Kidney	–
Diarrhea	Intestine	–
Dietary phytate	Intestine	–
Vitamins and Hormones		
Parathyroid hormone	Parathyroid	+
Calcitonin	Parafollicular cells of thyroid gland	–
Vitamin D	Liver, skin, intestine, and kidney	+
Bone Remodelling		
Osteoblasts	Bone	–
Osteoclasts	Bone	+

*+indicates a rise in serum calcium; –indicates a fall

Effects of Parathyroid Hormone

Parathyroid hormone raises serum calcium levels by elevating bone resorption, renal reabsorption of calcium, and synthesis of 1,25 dihydroxycholecalciferol.

Parathyroid hormone is responsible for maintaining adequate levels of serum calcium. The hormone is released from the parathyroid glands as the precursor hormone, proparathyroid hormone (proPTH, 94aa) or parathyroid hormone (PTH). Additional proteolytic modification of PTH takes place in various peripheral tissues. Release of PTH is stimulated by a fall in calcium or a rise in the antagonistic hormone, calcitonin, which will be discussed later. PTH functions by several mechanisms: it stimulates bone resorption, reabsorption of calcium in the renal tubules, and synthesis of dihydroxycholecalciferol [$1,25(OH)_2D_3$] which evaluates intestinal absorption of calcium and phosphate and itself stimulates bone resorption.

The effects of PTH on the kidney are likely mediated via adenylate cyclase-cyclic AMP but, in addition, require the presence of calcium. PTH has a very rapid

Table 21-5. Summary of the Roles of Parathyroid Hormone, Calcitonin, and Vitamin D on Bone Remodelling and Growth

Factor	Action	Direction
Parathyroid hormone	Bone resorption	+
	Renal Ca^{++} resorption	+
	1,25 $(OH)_2D_3$	+
	Osteoclast acid production	+
	Osteoblast collagen synthesis	–
	Renal phosphate excretion	+
	Osteoclast number	+
Calcitonin	Osteoclast number	–
	Organic acid synthesis	–
	Renal phosphate excretion	+
	Renal calcium excretion	+
Vitamin D	Intestinal calcium uptake	+
	Mobilization of bone calcium	+

effect in enhancing phosphaturia. In individuals with PTH-secreting tumors, there is a rise in blood calcium which cannot be effectively treated by diet. Treatment issues will be discussed later. The role of PTH is summarized in Table 21-5.

Effects of Calcitonin

Calcitonin is responsible for lowering serum calcium by stimulating bone growth, and calcium and phosphate excretion.

Calcitonin, which lowers serum calcium levels, is secreted from the parafollicular cells of the thyroid gland and is known as thyrocalcitonin in older literature. Support of the notion that calcitonin plays a role in overall calcium metabolism comes from the observation that people with calcitonin-secreting tumors have hyperplastic parathyroid glands. Further, calcitonin levels are affected by dietary calcium intake; the levels rise when a calcium-rich meal is fed after a fast. Young animals are apparently more sensitive to calcitonin than older animals, making it tempting to speculate that calcitonin insensitivity may play a role of osteoporosis. Calcitonin rapidly decreases the number of osteoclasts and the amounts of lactate and citrate produced by the osteocytes. It also increases the ability of the osteoblasts to synthesize collagen matrix necessary for mineralization. Paradoxically, calcitonin lowers the actual number of osteoblasts. This may explain why calcitonin is effective in Paget's disease, a pathologic bone resorption condition, for only a short period of time. Like PTH, calcitonin elevates urinary phosphate excretion and also increases calcium excretion. This is consistent with its lowering plasma calcium, but this part of its action counteracts its contribution to bone mineralization. The role of calcitonin is summarized in Table 21-5.

Effects of Vitamin D

Vitamin D has become a required nutrient because of our lifestyle. It is converted to its active form in a series of reactions to 1,25 dihydroxycholecalciferol [1,25$(OH)_2D_3$].

Vitamin D has been said to be a hormone which our lifestyle has made into a vitamin. Under conditions of adequate exposure to sunlight, vitamin D can be completely synthesized endogenously, travel to a target tissue via the bloodstream, and exert its actions in minute quantities—all of the qualifications of a hormone. The Recommended Dietary Allowance for vitamin D is shown in Table 21-6.

Table 21-6 Calcium, Phosphorus, and Vitamin D Recommended Dietary Allowances

	Age (yr)	Calcium (mg)	Phosphorus (mg)	Vitamin D* (μg)
Infants	0.0-0.5	360	240	10
	0.5-1.0	540	360	10
Children	1-3	800	800	10
	4-6	800	800	10
	7-10	800	800	10
Males	11-14	1200	1200	10
	15-18	1200	1200	10
	19-22	800	800	7.5
	23-50	800	800	5
	51+	800	800	5
Females	11-14	1200	1200	10
	15-18	1200	1200	10
	19-22	800	800	7.5
	23-50	800	800	5
	51+	800	800	5
Pregnant		+400	+400	+5
Lactating		+400	+400	+5

* As cholecalciferol — 10 μg cholecalciferol = 400 I.U. of vitamin D.

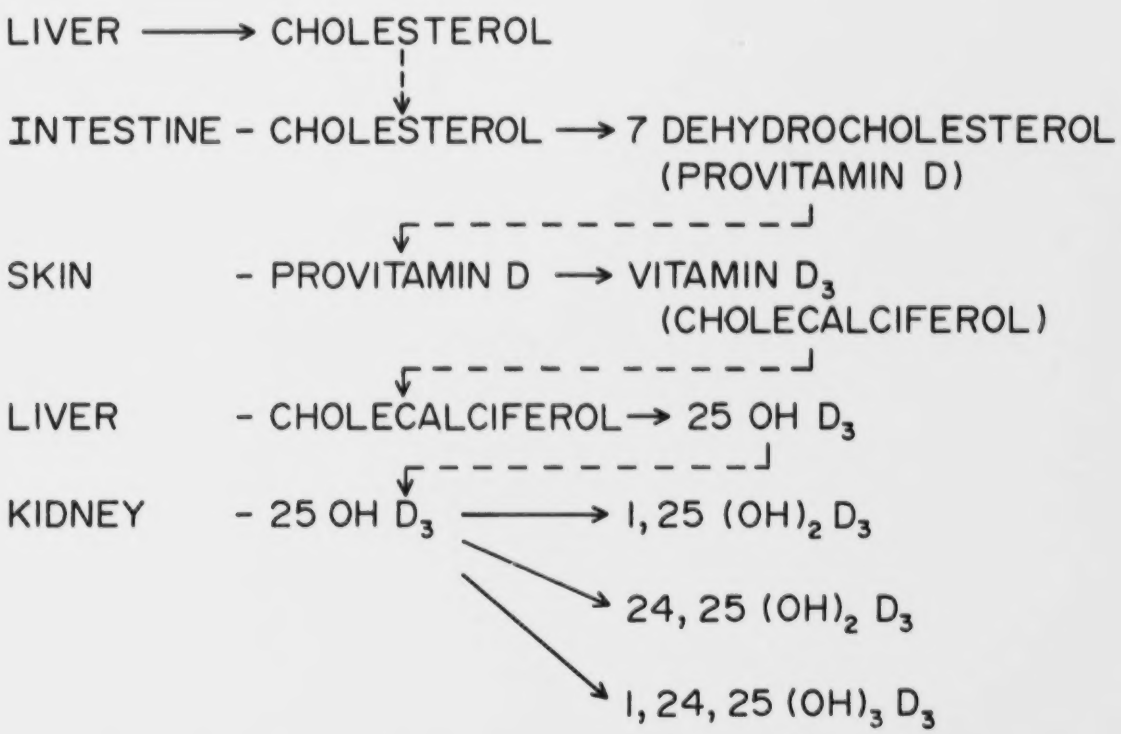
Adapted from Food Science and Nutrition Board, National Academy of Sciences—National Research Council: *Recommended Dietary Allowances*, Revised 1980.

Vitamin D is an excellent example of the interaction of a variety of tissues and regulatory mechanisms. The synthetic scheme for the active form of vitamin D is shown in Figure 21-1. Disease in any of the tissues involved in the synthesis and conversions of vitamin D should be expected to contribute to osteoporosis. For example, renal disease can prevent adequate formation of 1,25 $(OH)_2D_3$ or other metabolites, and diarrhea may prevent adequate calcium absorption, even in the presence of sufficient vitamin D. High plasma calcium concentrations inhibit the 1-hydroxylase enzyme, and PTH may be required for the synthesis of the 1-hydroxylase. Another vitamin D metabolite is also formed by the kidney — 24,25 dihydroxycholecalciferol. Only the 1,25 form stimulates uptake of calcium by the intestine, but both forms can stimulate mobilization of bone calcium. It is possible that the 24,25 hydroxylated form may be involved in feedback inhibition of PTH secretion. When there is adequate calcium, hydroxylation in the 24 position dominates; when calcium reserves are low, the 1 position is preferentially hydroxylated. There is a third metabolite, 1,24,25 $(OH)_3D_3$, whose role is still unclear. It may be a form of the vitamin marked for excretion by the liver. The role of vitamin D is summarized in Table 21-5.

Rickets in children and osteomalacia in adults can be prevented by adequate vitamin D and phosphate, or by 1,25 dihydroxycholecalciferol [1,25$(OH)_2D_3$]. Use of certain anticonvulsants or antibiotics may increase the risk of rickets.

Without adequate vitamin D, bone mineralization is incomplete, causing rickets in children or osteomalacia in adults. There is hypocalcemia and often secondary hyperparathyroidism. Bone matrix and cartilage syn-

Figure 21-1 Synthesis of Vitamin D and Its Metabolites


LIVER → CHOLESTEROL
INTESTINE - CHOLESTEROL → 7 DEHYDROCHOLESTEROL
(PROVITAMIN D)
SKIN - PROVITAMIN D → VITAMIN D_3
(CHOLECALCIFEROL)
LIVER - CHOLECALCIFEROL → 25 OH D_3
KIDNEY - 25 OH D_3 → 1, 25 $(OH)_2$ D_3
24, 25 $(OH)_2$ D_3
1, 24, 25 $(OH)_3$ D_3

theses are normal at first but eventually slow greatly in the absence of mineralization. Teeth which are formed during a period of rickets are abnormally calcified, leading to a greater incidence of caries in later life. Fortunately, vitamin D deficiency rickets is now rare in Western societies. There are, however, vitamin D-resistant forms of rickets which may respond to a combined treatment of vitamin D and phosphate supplementation or to the addition of 1,25 $(OH)_2D_3$. This may be accomplished with Calciferol, available in 50,000 USP unit tablets or 500,000 USP/cc in oil injections. This compound should not be given to patients with hypercalcemia or evidence of vitamin D toxicity, such as weakness, headache, vomiting, muscle or bone pain, loss of appetite, hypertension, or cardiac arrhythmias. Several types of phosphate supplements are available, but they may cause vomiting or diarrhea. In some cases, such as in children or adults receiving certain anticonvulsant drugs (e.g., phenytoin) osteomalacia may develop as the result of stimulation of the enzyme system responsible for vitamin D breakdown. These drugs should be used in the lowest effective dosages possible and for the shortest time periods needed to produce therapeutic effects. Certain antibiotics, such as tetracycline, may tie up calcium in the gastrointestinal tract. Chronic renal disease may also precipitate bone loss as a consequence of failure to hydroxylate 25$(OH)D_3$ to 1,25$(OH)_2D_3$ in the kidney. There may be build-ups of the 25$(OH)D_3$ form or other vitamin D metabolites which themselves may interfere with normal calcium homeostasis. Likewise, liver disorders may result in a lack of sufficient hydroxylation in the 25 position to form the 25(OH) intermediate. There are some conditions which may be confused with rickets.[7] Usually, however, there is some method for differentiating other disorders from rickets (see Table 21-7).

Table 21-7. Conditions that May Be Confused with Rickets

Condition	Differentiation from rickets
Intrinsic bone disease	
Achondroplasia	Normal serum chemistries Radiological determination
Metaphyseal chondroplasia	
(Jansen type)	Hypercalcemia
(Schmid type)	No hypercalcemia
Genetic mucopolysaccharidoses	
Morquio's syndrome	Urinary mucopolysaccharide
Hurler's syndrome	Urinary mucopolysaccharide
Hormonal disorders	
Syndromes involving pituitary, thyroid, adrenals, gonads	Growth plates well-defined
Hyperparathyroidism	Chronic elevated PTH, late childhood, normal or accelerated growth; hips and ankles more affected than knees or wrists; long-bone bowing uncommon
Vitamin C deficiency	Fractures in metaphysis produce a zone of compressed, disorganized calcified cartilage resembling healed rickets
Metaphyseal defects	
Hypophosphatasia	Prominent cupping deformity Phosphorylethanolamine in urine
Osteogenesis imperfecta	Defective formation of primary and secondary spongiosa
Osteopetrosis	Bone-in-bone appearance of radiographs; less space for bone marrow
Localized disorders	
Blount's disease	No widening of growth plate at proximal end of tibia
Trauma, Radiation, Frostbite, Myelodysplasia	Sometimes difficult to differentiate from rickets; examine multiple bone sites

Adapted from Reference 7.

Effects of Insulin

Insulin insufficiency leads to generalized growth retardation. Bone demineralization may occur in diabetic metabolic acidosis.

Another group of hormones which affects bone metabolism are those hormones which ordinarily regulate nutrient homeostasis.[6] Among these are insulin, growth hormone, thyroid hormone, the sex steroids, and glucocorticoids. Some of the actions of these hormones on bone may be secondary or tertiary to their impact on other target tissues, and certainly many of their interactions with bone are not well understood. For example, in insulin deficiency in children, there is generalized growth retardation, but it is not clear whether the effect is one of generalized nutrient deficiency or whether there are more direct actions on bone cells. Since insulin is known to alter intracellular calcium pools in some cells, it is also possible that insulin could affect bone calcium distribution in a direct way. Bone demineralization may occur during periods of metabolic acidosis in diabetics. The goal for such patients should be to prevent ketosis and acidosis by maintaining good blood glucose control. (See the Guide Index for other modules which outline dietary strategies for the diabetic.)

Effects of Growth Hormone

Growth hormone is necessary for bone growth, and insufficiency leads to reduced stature and perhaps to sexual immaturity. Excesses are equally detrimental, producing gigantism in the young and acromegaly in adults.

Effects of growth hormone on bone are direct, producing dose responses in bone growth. Lack of the hormone during developmental periods leads to a failure to achieve full stature (dwarfism), while excesses during this period result in abnormal excess growth (gigantism). Pituitary dwarfism is the result of the failure of the pituitary to produce growth hormone, either as part of an overall lack of hormones from the pituitary (panhypopituitarism) or as a particular deficiency of growth hormone (sexual ateliotic dwarfism). Those with panhypopituitarism will not mature sexually and will show signs of hypothyroidism and adrenal insufficiency. Lack of only growth hormone, however, impairs attainment of full stature and not sexual maturity. After fusion of the epiphyseal plates, additional bone growth is limited to areas of responsive cartilage such as the jaw, hands, and feet, leading to the characteristic deformities of acromegaly. The effects of growth hormone on bone are apparently mostly on calcification and chondrogenesis without speeding up the bone maturation process and the fusion of the epiphyseal plates. It is generally believed that growth hormone exerts its actions on bone via the somatomedins, among which is insulin-like growth factor I.

Effects of Thyroid Hormone

Hypothyroidism slows bone turnover while hyperthyroidism increases bone turnover and leads to negative calcium and phosphate balances.

Thyroid hormone appears to have dramatic interactions with bone. In hypothyroidism bone turnover is slowed, while in hyperthyroidism it is increased. During development, hypothyroidism leads to impairment of skeletal growth. Bone resorption by osteoclasts has been shown to occur *in vitro* in response to thyroid hormone. There are associated increases in serum calcium and a decrease in PTH and 1,25 $(OH)_2D_3$, as would be expected. Cartilage growth may be stimulated. Unlike growth hormone, thyroid hormone promotes bone maturation. Excesses of thyroid hormone lead to negative calcium and phosphate balances and to decreased absorption from the intestine. Hyperthyroid patients should be monitored closely to determine that they are receiving adequate dietary calcium.

Effects of Sex Hormone

Androgens and estrogens are involved in bone maturation and fusion of the epiphyseal plates.

Clearly, the sex steroids (androgen and estrogens) must also be involved in skeletal maturation and maintenance. Skeletal size of males is usually greater than

females, while the female skeleton provides a necessary reserve of calcium to support pregnancy and lactation. Further, in postmenopausal women there is a dramatic loss of bone mass which can be slowed by estrogen supplementation (discussed in greater detail in the section "Treatment of Osteoporosis").

Premature secretion of androgens or estrogens results in early fusion of the epiphyseal plates. The mechanisms of action of these hormones are unknown. It has been postulated that part of the androgen effect is due secondarily to increased muscle mass, which seems to favor increased bone mass. Other effects may be mediated by changes in calcium absorption from the intestine, alterations in 1,25 $(OH)_2D_3$ synthesis, or stimulation of calcitonin secretion in order to inhibit bone resorption. Androgens have been used to promote bone healing in aging patients, but their effects are short-term. Besides producing virilism in female patients, androgens may cause hepatic dysfunction. Estrogen will be discussed later in this module.

Effects of Glucocorticoids

Glucocorticoid effects on bone are beneficial at physiological concentrations but detrimental at high concentrations.

The effects of glucocorticoids depend upon their concentrations; i.e., whether they are present in physiological or pharmacological amounts. In excess, the glucocorticoids diminish calcium and phosphate uptake from the intestine and increase their excretion from the kidney. There is increased bone turnover and a precipitous drop in the number of osteoblasts at sites of new bone formation resulting in glucocorticoid-induced osteoporosis. Patients receiving glucocorticoid therapy for adult respiratory distress syndrome, organ transplants, or neoplastic diseases may all experience increased bone turnover as a result of the high glucocorticoid levels. At normal concentrations, however, the glucocorticoids have beneficial effects on skeletal growth. At normal concentrations, there is actually a period of stimulation of bone collagen synthesis by glucocorticoids in bone cultures. Glucocorticoid effects may be mediated by regulation of differentiation of the various types of bone cells. Patients receiving drugs such as prednisone may need to follow a low-sodium, low-simple-carbohydrate diet and try to avoid gaining weight. When pharmacological doses are necessary for a long course of therapy, calcium supplementation may to be in order.

Effects of Paracrine Factors

Bone itself produces factors which, acting locally, alter its own metabolism.

The final group of hormones/factors which help to regulate bone formation and turnover may operate in a paracrine rather than a hormonal fashion. Prostaglandins (PGE series) are potent stimulators of bone resorption. In some bone tumors, there is an elevated production of prostaglandins, which leads to hypercalcemia and considerable resorption in the region of the tumor.

Factors in blood cells may play a role in the regulation of bone metabolism. One of these is the osteoclast-activating factor derived from proliferating lymphocytes. *In vitro* it stimulates bone resorption and inhibits collagen synthesis. This may account for the hypercalcemia associated with such diseases as myeloma. Mithramycin IV (Mithracin), an antineoplastic agent, may be considered for treating such patients in a hospital setting. With such treatment, calcium balance may return to normal rather quickly. Treatment by low dietary calcium is not recommended.

Since bone remodelling occurs in response to mechanical stress, a local phenomenon, local factors must exist to mediate the changes. These factors may be related also to the somatomedins. One factor, bone growth factor II, has been reported to stimulate synthesis of new cells and matrix in cartilage cultures.[6] Although these factors may be derived from other tissues as well, current data seem to indicate that they can arise from cells in the bone. A great deal of additional research must occur before the roles of these factors are clarified.

Clearly, bone growth and remodelling is a complicated process involving not only bone tissue itself but also many other body tissues. Impairment in any of these other tissues may be expected to cause anomalies in the turnover of bone. If a severe enough imbalance is created, then osteoporosis will be the ultimate result.

Risk Factors For Osteoporosis

Although osteoporosis may be a natural consequence of aging, the degree of osteoporosis is a function of peak bone mass attained in early adulthood.

Much of the osteoporosis commonly seen is age-related, but must it be an inevitable part of the aging process? Evidence indicates that bone loss, particularly in women, begins in the third decade of life, although increased fracture risk may not become apparent until ages 60-70. If bone loss occurs as early as age 30, perhaps there are some preventive measures which can be employed to delay the onset of loss or to avoid it entirely. It is generally believed that the degree of subsequent bone loss is a function of peak bone mass attained in early adulthood. Prudence would seem to indicate that actions should be taken to ensure reaching the genetically determined maximum bone mass. The ideal time to discuss the prevention of osteoporosis with female patients is during the yearly female exam.

Postmenopausal women of western European extraction most often develop osteoporosis. Also at risk are women who have had an oophorectomy at an early age, women who have excessive alcohol intake, cigarette smokers, and those who lead a sedentary life style.

Seemingly at greatest risk of osteoporosis are postmenopausal, fair-complexioned women of western European descent. Approximately 15% of the postmenopausal women in the U.S. will develop osteoporosis. The incidence in black women is very much lower.[8] These data would seem to indicate a genetic component for the disease which is separate from nutritional considerations. There does, however, seem to be a correlation of development of osteoporosis with lowered calcium intake. National dietary surveys have indicated that 66% of U.S. women have calcium intakes below the RDA in the years when peak bone mass is attained, and that 75% of older women have inadequate calcium intakes. Blacks generally have lower calcium intakes than whites. In comparing osteoporotic and non-osteoporotic women of the same ages, the osteoporotic groups had lower calcium intakes during a period of time prior to the diagnosis of osteoporosis. Retrospective studies have indicated that excessive alcohol intake, cigarette smoking, and sedentary lifestyles are associated directly or indirectly with osteoporosis in later life.[9] While no causality can be implied by these studies, it is not difficult to suppose, for example, that excess alcohol could cause liver or intestinal damage, which in turn might interfere with vitamin D metabolism and calcium uptake. Other factors known to be capable of precipitating osteoporosis are the chronic use of anticonvulsant drugs, such as phenytoin, and long-term antacid abuse leading to hypophosphatemia.

Prevention of Osteoporosis

Dietary Calcium

Dietary calcium is best supplied by dairy products; but for reasons of economics, food preferences, or intolerances, it may be difficult for many people to obtain an adequate amount of calcium from dietary sources.

The best ways to prevent osteoporosis and to treat the disease, once it is evident, are controversial. A number of studies indicate life-long patterns of less-than-adequate calcium intake resulting in osteoporosis. In general, men consume nearly twice as much calcium as women, and women have the added drain on their calcium supplies of pregnancy and lactation.[10] The recommended dietary allowances for calcium, phosphate, and vitamin D for different age groups are given in Table 21-6. During pregnancy, there is elevated calcium absorption and perhaps even positive calcium balance, with the additional calcium being deposited in the mother's bones. During lactation, there is an additional requirement for calcium and phosphorus because of the losses of these constituents to the milk. Calcium balance during periods of lactation is usually negative.

Intake of calcium is also a function of economic level, since foods that contain the largest amounts of calcium tend to be those which are more expensive. Concerns about calorie intake, ethnic eating patterns, or food intolerance may prevent some individuals from receiving adequate calcium. Food choices of various ethnic groups were discussed in Module 1, Nutrient Content of Foods, Nutritional Supplements, and Food Fallacies.

In older people there may be a loss of appetite or strong food dislikes which can impinge on food choices.

Many older people think milk is only for babies; others may think dairy products are "harmful" because of the cholesterol content. Still others think that cheese causes constipation. Lactose intolerance can contribute to lowered intakes of high-calcium dairy products, but lactose intolerance is found most often in oriental and black populations who are not at particular risk for osteoporosis. Even in lactose-intolerant persons, adequate calcium can be obtained by ingestion of fermented dairy products. The calcium contents of some common foods are shown in Table 21-8. Pasteurization or homogenization of milk does not lower the availability of calcium. Calcium availability is reduced in foods with high oxalate levels (soybeans, kale, etc.) and in foods

Table 21-8. Calcium Content of Common Foods

ITEM	SERVING SIZE	CALCIUM CONTENT (mg)
Milk, skim, whole, 2%	8 oz	300
Milkshake, homemade	8 oz	360
Milk, malted	8 oz	345
Milk, chocolate or buttermilk	8 oz	285
Swiss cheese	2 oz	550
Cheddar cheese	2 oz	410
Colby cheese	2 oz	390
Processed American cheese	2 oz	350
Cottage cheese	½ cup	75
Yogurt, plain, lowfat	8 oz	415
Ice cream	1 cup	170
Pudding	½ cup	135
Pizza, with cheese	1 medium piece	145
Macaroni and cheese	1 cup	360
Sardines, canned	2 oz	170
Salmon, canned, with bones	2 oz	150
Oysters, raw	7 - 9	115
Collards, raw	½ cup	180
Mustard greens or kale, raw	½ cup	100
Blackstrap molasses	1 Tbsp	135
Dried beans, cooked	1 cup	90

Adapted from Module 8, Normal Diet: Geriatrics.

with high phosphate levels, such as unpolished rice and bran. A general rule of thumb is to divide the amount of calcium in foods from these sources by 2 to get the quantity of calcium actually available for absorption.

Dietary Phosphorus

In animal studies, the Ca/P ratio has been found to be important to normal growth and maintenance of the skeleton. It has been suggested that a Ca/P ratio of 1, or a little greater, should be optimal in humans.

Along with dietary calcium intake, phosphorus intake must be taken into consideration. In animals it has been shown that excess phosphorus or low amounts of dietary calcium relative to phosphorus result in skeletal defects. In humans, however, there is little evidence that this is the case and even some evidence that calcium balance may be improved by elevated phosphorus intakes. There is an inverse relationship between dietary phosphorus and urinary calcium. This results in elevated PTH and increased bone resorption. It has been suggested that a Ca/P ratio of 1, or a little greater, would be ideal, but this is difficult to achieve by dietary means alone. In the U.S., consumption of phosphate is rising. Cola drinks contain large amounts of phosphate (12-20 mg%). Phosphates, including orthophosphate (for acidification), and di- and polyphosphates are frequently used as food additives.[4] Dairy products have only marginally more calcium than phosphate. Processed cheese, on the other hand, actually has more phosphate than calcium. Meat which is rich in phosphorus is consumed 31% more now than 20 years ago, while dairy product consumption is down 37%. It has been shown[11] that lacto-ovo vegetarians have a greater bone mass than omnivores. Since meat is high in phosphates and since this type of vegetarian uses dairy products having a Ca/P ratio of about 1, this lends credence to the hypothesis that a balance between calcium and phosphate is beneficial. Most of the dietary phosphorus is absorbed as free phosphate. With normal intakes of phosphorus, absorption efficiency is 60-70%; this may be lowered when a significant amount of dietary phosphate is obtained from cereals and seeds which contain phytate, an organic ester of phosphate which is not readily digestible (see also the section "Drugs and Calcium Absorption").

Age and Calcium Absorption

In young people who are still growing, the efficiency of dietary calcium absorption is 75%, but it drops to 30-60% in adults.

Calcium absorption declines with age, so an additional complicating factor is involved. In actively growing children, about 75% of the available dietary calcium is absorbed; in adults on a normal diet, this figure may drop to 30-60%. Thus, even in calcium-adequate diets there may be a reduced uptake of available calcium. Some of the reduction in absorption may be due to reduced circulating levels of $1,25(OH)_2D_3$. It has been postulated that there is a mild, compensatory parathyroid-mediated bone loss with aging. Other hypotheses suggest that overall bone loss is a function of imbalance between formation and resorption of bone caused by physical inactivity or steroid deficiency.

Another contributing factor to decreased calcium absorption may be the amount of dietary fiber. Although believed to have beneficial effects on lowering serum lipid levels and preventing gastrointestinal tract problems, fiber can effectively bind minerals such as calcium. For a review, see the recent work by Heany et al.[10] Long-term studies have not been performed to determine whether the fiber content of diets might have serious consequences for calcium balance. The apparent mechanism for the bone loss would dictate the most efficacious treatment. Total vegetarians may have lowered bone mass (as opposed to lacto-ovo vegetarians who have been shown to have elevated bone mass),[11] and may refuse calcium supplements as "unnatural." Their rigid exclusion of dairy products from the diet may be extended to their children as well. These patients should be counseled about the short- and long-term hazards of these dietary practices, both for themselves and especially for their children. Fortunately, it is relatively easy for an adolescent to meet the RDA for calcium because foods such as cheese pizza, cheeseburgers, yogurt, milkshakes, etc. find high acceptability in this age group.

Drugs and Calcium Absorption

Laxatives and antacid abuse are common causes of poor calcium absorption.

There are a number of commonly used prescription and over-the-counter preparations which may interfere with calcium uptake or metabolism. As mentioned previously, antacid (aluminum hydroxide-containing antacids such as Maalox, Mylanta, Amphojel, and Gelusil) abuse may lead to phosphorus depletion, which in turn could precipitate osteomalacia. The condition is reversible when the abuse ceases and phosphorus is repleted. Heavy antacid use may be the result of self-medication, but it is also seen in patients with ulcers or in those receiving glucocorticoid when it is administered to reduce the risk of ulcers.[12] The practice of taking these antacids shortly after eating undoubtedly reduces phosphorus availability. Furthermore, much of the dietary phosphorus may be present as phytate, which is available only after hydrolysis. The activity of the intestinal enzyme (phytase) responsible for this reaction is believed to be dependent upon vitamin D. Other drugs, such as cellulose phosphate, an ion exchange resin, bind calcium itself. Similar findings have been reported for cholestyramine resins. Long-term use of colestipol, a bile acid sequestrant, may also change serum calcium levels, perhaps by altering vitamin D absorption. Since calcium is more readily absorbed in an acidic rather than in an alkaline environment, patients with achlorhydria also have lowered calcium absorption.

Laxative abuse may limit calcium absorption, perhaps by decreasing transit time of nutrients in the gut. Phenolphthalein, a common laxative, has been reported to be associated with osteomalacia.[12] Drugs which have a toxic effect on the mucosal cell lining would all be expected to have effects of varying severity on calcium and phosphate uptake, depending upon the degree of damage to the mucosal lining. The drugs which interfere with calcium absorption are summarized in Table 21-9. For patients receiving these treatments, adequate calcium intake should be ensured. If dietary intake is inadequate, then supplements should be used.

Calcium can have an adverse effect on the absorption of certain drugs as well. One of the most notable cases is tetracycline, which combines with calcium to form a nonabsorbable complex. Further, use of tetracycline during the last half of pregnancy, infancy, and childhood (to age 8) may result in a child's having permanently mottled teeth that are extremely susceptible to decay. Some antibiotics such as penicillin, chloramphenicol, and neomycin, however, actually increase absorption of dietary calcium.

Table 21-9. Drugs Which Interfere With Calcium Absorption

Aluminum-containing antacids
Maalox
Mylanta
Amphojel
Gelusil
Exchange resins
Cellulose phosphate
Cholestyramine
Colestipol
Anticonvulsants
Phenytoin
Antibiotics
Tetracycline
Laxatives
Phenophthalein

Treatment of Osteoporosis

Calcium Supplementation

Calcium from several types of supplements is readily available, but calcium carbonate supplements provide the highest percentage of calcium, by weight.

The easiest way to provide additional calcium, besides rational food choices, is by exogenous supplementation. Not all supplements provide the same amount of calcium by weight. The amount of calcium in various preparations is shown in Table 21-10.

Calcium carbonate provides the highest percentage of calcium per weight of pill. This may be an important consideration when there is a need to provide considerable supplemental calcium. All of the preparations are about equally absorbable and comparable to the absorption of calcium from milk. Dairy products have the advantage, however, of having an optimal ratio of Ca/P. These supplements should be taken throughout the day along with a small amount of milk or yogurt, both of which contain vitamin D and lactose which promote the absorption of calcium from the gastrointestinal tract.

Table 21-10. Calcium Content of Common Supplements

Preparation	% of Calcium (by wt)	Note
Calcium carbonate	40%	Contained in antacids such as Tums
Calcium phosphate	31%	See bone meal
Calcium lactate	13%	Poor absorption
Calcium gluconate	9%	Poor absorption
Bone meal	31%	Contains other elements and may be contaminated with toxic metals
Dolamite	22%	Ingredient in several antacids; could be contaminated with toxic metals

At levels of calcium ingestion of greater than 2.5 gm/day, there may be calcification of soft tissues, hypercalciuria, hypercalcemia, and formation of urinary calculi.

Calcium does not appear to be toxic in the concentrations likely to be ingested, but it is possible to consume enough to produce side effects. These include hypercalcemia; hypercalciuria; calcification of the soft tissues, including the kidney and artery walls; formation of urinary calculi; and supression of bone remodelling. Under normal circumstances, these would not be a problem until consumption is greater than 2.5 gm/day. In patients on chronic renal dialysis, however, normal calcium homeostasis is greatly impaired; so strict attention must be paid to calcium balance. Vitamin D may be given to enhance calcium utilization. Another group of patients at risk are those with ulcers who drink large quantities of milk and consume similarly large quantities of antacids, not a recommended treatment modality. They may develop milk alkali syndrome which presents itself as hypercalcemia and alkalosis.

Fluoride Supplementation

Treatment of osteoporosis with combined fluoride and calcium therapy has proved beneficial.

Fluoride use has been suggested because it stimulates bone formation and has been reported to increase bone mass in osteoporotic patients.[13] Fluoride is incorporated into bone as a substitute for the hydroxyl groups in hydroxyapatite. Levels of 50mg NaF/day, plus 1 gm of calcium as calcium carbonate, have been used therapeutically. Some clinicians recommend lower doses for better tolerance. Sodium fluoride is available as a prescription. Ongoing studies and various biases do not allow us to recommend one standard dose at this time. Further, some epidemiological evidence[14] supports the notion that adequate fluoride intake reduces the incidence of osteoporosis. Since fluoride alone may cause defective bone mineralization, it has been suggested that calcium supplementation be an integral part of fluoride therapy. In some cases, fluoride therapy has been associated with rheumatic symptoms (joint pain and swelling), gastrointestinal symptoms (nausea, vomiting, blood loss anemia, and ulcers), or combinations of both. The amount of fluoride which is absorbable depends upon the degree of hardness of the local water supply. Hard water leads to the formation of insoluble calcium-fluoride complexes, thereby lowering fluoride absorption. Supplemental fluoride is currently recommended only in the therapy of osteoporosis, not in prevention. The average diet contains 0.2 - 0.6 mg fluoride/day.[14] Diets high in seafoods or tea provide additional amounts. Fluoridated water is the best source of fluoride and is completely safe in the amounts currently added to the water supply.

Fluoridated water consumed during the period before tooth eruption provides the greatest protection against dental caries.

It has long been known that fluoride consumed during tooth development provides protection against dental caries once the teeth have erupted. Fluoride concentrations in the water of greater than 1 ppm produce this protective effect. Excessive fluoride (greater than 3 ppm in water) consumed during periods of tooth development, however, results in a mottled appearance of the tooth enamel. Topical fluoride applied after the teeth have erupted is less effective. Since developed teeth have limited ability to repair themselves and virtually no communication with systemic circulation, the environment of the mouth is more important in considering any potential effects of topical or ingested fluoride. It may be possible, however, that fluoride interferes with the metabolism of the oral bacteria rendering them less cariogenic.

Vitamin D Supplementation

If vitamin D is used in the treatment of osteoporosis, it should be done with caution because of the potential toxic effects.

Vitamin D status obviously impacts on osteoporosis treatment. Dietary sources of the vitamin are found naturally in oily fish, eggs, liver, and fortified products such as milk, which is required by law to have 10μg/qt (400 IU/qt). The average daily intake in the U.S. is between 4 and 6μg. Studies of the vitamin D status in the older segments of the population are sketchy.[15] Requirements in the elderly have not been clearly defined. Some studies have been unable to show a beneficial effect of vitamin D supplementation on reducing osteoporotic fractures. Vitamin D is stored in fatty tissue and can build up to toxic levels, resulting in elevated serum calcium and nephrocalcinosis. This led investigators to suggest that since vitamin D supplements could result in toxic doses and since hypercalcemia and hypercalciuria have been observed with vitamin D supplementation, vitamin D should not be used therapeutically.[10] Other practitioners include vitamin D (400-800 IU/day) in a combined treatment regimen with fluoride (0.1 mg/kg bodyweight) and calcium (1500 mg/day).[16] If a vitamin D supplement is used, it would seem wise to use a dose similar to the RDA, which is 400 IU/day.

Estrogen Supplementation

Estrogen therapy for osteoporosis should be used only with caution and is most effective immediately after the onset of menopause.

Estrogen therapy begun soon after the onset of menopause slows bone loss; therapy begun later is less effective. It has been reported that 1,25 $(OH)_2D_3$ and calcitonin are increased in estrogen therapy. Estrogen may act on osteoclastic precursors in bone by raising the threshhold for PTH sensitivity. As summarized by Saville,[17] studies reported that 0.625 mg/day of conjugated equine estrogen, 1 mg/day of estradiol-17B, or 0.15 mg/day ethynyl estradiol are effective and minimize side effects. Estrogen therapy itself carries some additional risks.[18] One of these is a 3-7 times greater risk of endometrial cancer. Use of progestins in a cyclic treatment (at least for 10 days/month) for patients with an intact uterus may ameliorate some of the risk of estrogen use.

Use of estrogen therapy entails some special precautions: patients must be closely monitored for symptoms of breast and uterine cancer, and the smallest doses of estrogen possible should be used. The finding that additional calcium (1 gm/day) compensates for the lack of estrogen[17] makes the use of estrogen supplementation a matter for considerable deliberation. Women undergoing oophorectomy at early ages would be candidates for estrogen therapy, as would thin, white women with multiple risk factors present for osteoporosis. All women receiving or contemplating the use of estrogen should be counseled about the relative risks involved with estrogen use.

Exercise is an important component of maintenance of healthy bones. Patients should be encouraged to continue moderate exercise throughout their lives.

Finally, the effects of exercise must be considered. It has long been known that bone mass decreases with immobilization. This has been further substantiated by

investigations into the effects of weightlessness in space-flight studies.[19,20] Exercise may not only slow bone loss rates but may actually increase bone mass, providing there is adequate dietary calcium available. There does seem to be an optimal level of exercise. A recent report[21] has indicated that women joggers who trained with sufficient intensity to become amenorrheic also suffered bone demineralization, even though the average age of the women was only 25. It seems appropriate to counsel patients that walking and swimming in amounts that promote cardiovascular fitness are desirable. Even bed-ridden patients should be encouraged to do whatever isometric exercises are possible, taking into account their particular physical limitations.

Summary

Osteoporosis is the loss of bone mass resulting in increased fracture risk, particularly of the wrist, hip, and spine. Postmenopausal, white women of European extraction are at the greatest risk. Since the degree of risk is related to the peak bone mass attained early in adulthoood, the most effective measures are aimed at providing adequate calcium nutriture and plenty of exercise during this time period. Once osteoporosis is evident, there is little chance of reversal. Calcium supplementation alone or with combinations of fluoride and/or vitamin D have been used with some success. Estrogen therapy, which entails some additional risks, may also be used. Exercise is very important in arresting further bone loss in this population group. A chart of appropriate follow-up questions for different patient age groups is included to aid in monitoring information exchange with the patient (Table 21-11).

Table 21-11. Patient Information Exchange for Optimal Bone and Teeth Health

	Patient Age (Years)								
Exam types	0-10	10-20	20-30	30-40	40-50	50-60	60-70	70-80	>80
Questions	Well Baby School	School Employment	Employment Pap Smears Insurance	Employment Pap Smears Insurance	Employment Pap Smears Insurance	Employment Pap Smears Insurance	Employment Pap Smears Insurance	Pap Smears Routine	Pap Smears Routine
1. Fluoridated H_2O	X	X	X	X	X	X			
2. Topical fluoride		X	X	X	X	X	X		
3. Calcium intake	X	X	X	X	X	X	X	X	X
4. Exercise	X	X	X	X	X	X	X	X	X
5. Weight advice	X	X	X	X	X	X	X	X	X
6. Laxative abuse		X	X		X	X	X	X	X
7. Antacid abuse				X	X	X	X	X	X
8. Stature (ht.)	X	X	X	X	X	X	X	X	
9. Estrogen supplements					X	X			
10. Fractures					X	X	X	X	X
11. Vitamin status (dietary and supplements, especially vitamin D)	X	X	X	X	X	X	X	X	X
12. Glucose tolerance (diabetes)				X	X	X	X	X	X

Evaluation

Evaluation in this module is approached factually. Awareness of the problems associated with the maintenance of healthy bones and teeth is the key to successfully managing this component of your patients' health. Answer the following questions, and check your answers by referring to the appropriate section.

1. When should measures be initiated to prevent or retard the effects of osteoporosis?
2. What type of bone is affected by osteoporosis, and where is this bone found?
3. If a child is seen with massive decay of the upper front teeth, to what may the problem be attributed?
4. How is vitamin D related to bone growth and development?
5. What are the risk factors for osteoporosis?
6. What steps can be taken to prevent osteoporosis?
7. How can osteoporosis be treated?

If you have competed the above questions and wish to further test the knowledge you obtained from the module, do the following:

1. Select a patient from your practice who is at risk for osteoporosis.
2. Design a preventive and/or treatment plan for the patient. Include in your own plan both the medical management of the patient and what patient education you would conduct.
3. Present your patient and your plan to fellow physicians, faculty, and nutrition specialists. Discuss its strengths and weaknesses.

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Administration on Aging
Office of Human Development
U.S. Dept. of Health and Human Services
Washington, D.C. 20201

American Dietetic Association
Publication Department
620 N. Michigan Ave.
Chicago, Illinois 60611

American Medical Association
535 N. Dearborn St.
Chicago, Illinois 60610

Building Sound Bones and Muscles
O.E. Allen, Editor
Library of Health (1980)
Time-Life Books
Alexandria, VA 22310

Extension Service/Home Economics
U.S. Dept. of Agriculture
Washington, D.C. 20250
(or see your local county extension agent)

Pamphlet Series on Calcium
National Dairy Council (1984)
6300 N. River Rd.
Rosemont, Illinois 60018

J. Brody
"Calcium, the Vital Mineral"
Family Circle 3/6, 62-65, 1984.

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Stand Tall! The Informed Woman's Guide to Preventing Osteoporosis.
Triad Publishing Co. (1984)
Gainesville, FL 32610

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Some Abbreviations Used in the Nutrition in Primary Care Series

ATP	adenosine triphosphate
c	cup
cc	cubic centimeter
CNS	central nervous system
FDA	Food and Drug Administration
gm	gram
IBW	ideal body weight
IU	International Units
kcal	kilocalorie
kg	kilogram
lb	pound
lg	large
MCV	mean corpuscular volume
MDR	minimum daily requirement
med	medium
μg	microgram
mEq	milliequivalent
mg	milligram
MJ	megajoule
ml	milliliter
oz	ounce
RDA	Recommended Dietary Allowances
RE	retinol equivalents
sl	slice
sm	small
Tbsp	Tablespoon
TPN	total parenteral nutrition
tsp	teaspoon
USDA	United States Department of Agriculture